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CASE REPORT

CLINICAL CASE

Concurrent Acute Ischemic Stroke and Myocardial Infarction Associated With Atrial Fibrillation

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ABSTRACT

An 85-year-old man was admitted with dysarthria. Electrocardiography showed atrial fibrillation and prominent ST-segment elevation in V_2-V_6. Multiple acute cerebral infarctions were observed in brain images. Coronary angiography showed total occlusion of the mid left anterior descending artery. After thrombus aspiration, no atherosclerotic changes were observed on intravascular ultrasound. (J Am Coll Cardiol Case Rep 2024;29:102145) © 2024 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

An 85-year-old man visited the emergency department with a sudden onset of dysarthria in the morning. He was confirmed to be normal the night before, but his family noticed his dysarthria in the morning. His symptoms did not improve, so his family took him to the emergency department 7.5 hours after his symptoms were first noticed. Communication was difficult due to dysarthria, and the patient was unable to respond properly to commands. The NIHSS (National Institutes of Health Stroke Scale) score was 7 (1b, 2; language, 3; dysarthria 2). On neurologic examination, except for dysarthria, cranial nerve examination, motor and sensory grade, and deep tendon reflex were normal. No cardiac murmur was detected.

LEARNING OBJECTIVES

- To be able to make early and precise diagnosis of cerebral and myocardial infarctions.
- To understand possible causes of concurrent cerebral and myocardial infarctions.

PAST MEDICAL HISTORY

The patient had a medical history of hypertension, hyperlipidemia, and a previous stroke that resulted in right-sided weakness. He had been taking both aspirin and clopidogrel, as well as atorvastatin 10 mg as a treatment for a previous cerebral infarction that occurred 5 years ago. He had never been diagnosed with atrial fibrillation (AF) and had never taken anticoagulants.

DIFFERENTIAL DIAGNOSIS

The potential cause of dysarthria might include cerebral infarction, cerebral hemorrhage, and brain tumors.

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ABBREVIATIONS AND ACRONYMS

AF = atrial fibrillation

CCI = cardiocerebral infarction

MI = myocardial infarction

MCA = middle cerebral artery

STEMI = ST-segment elevation myocardial infarction

INVESTIGATIONS

His blood pressure was 149/79 mm Hg, and his pulse rate was 112 beats/min. Newly noted AF and prominent ST-segment elevation was observed between V_2 and V_6 on electrocardiography (Figure 1). When ST-segment elevation was confirmed, the patient had no complaint of chest pain. The serum high-sensitive troponin-I level was 83.3 ng/L, and the creatine kinase-myocardial band level was 1.7 mg/mL. Brain computed tomography angiography and magnetic resonance imaging were performed, and multiple acute cerebral infarctions were confirmed in both middle cerebral artery (MCA) and right posterior inferior cerebellar artery (Figure 2).

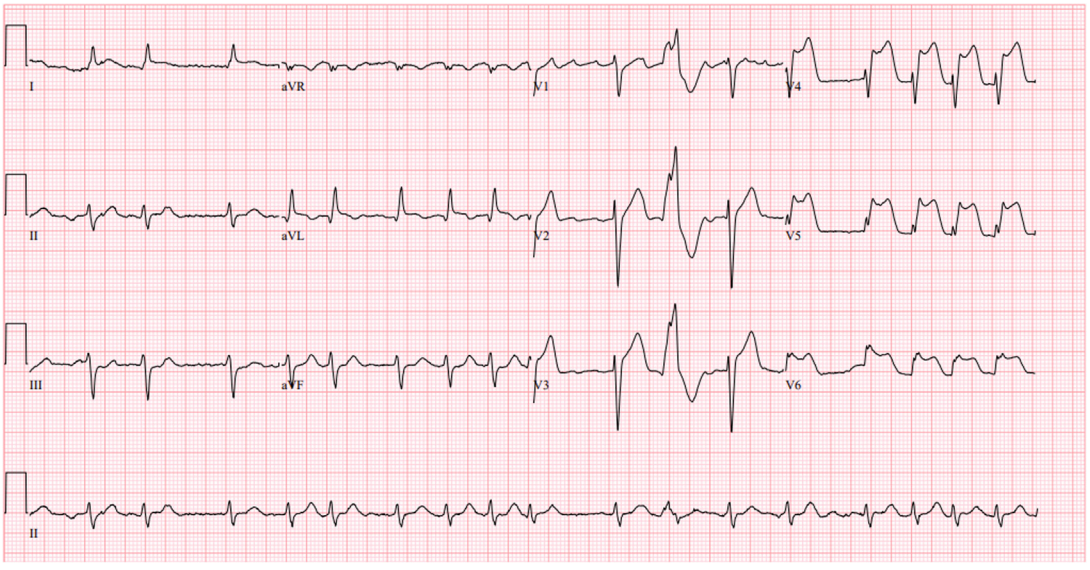
MANAGEMENT

Because the patient arrived at the hospital 7.5 hours after symptoms onset, fibrinolytic therapy was not indicated for cerebral infarction. The patient gestured chest pain 1 hour after the ST-segment elevation was confirmed and 2 hours and 30 minutes after visiting the emergency department. Coronary angiography was performed as a first-line therapy for ST-segment elevation myocardial infarction (STEMI) and showed total occlusion of mid left anterior descending artery (Figure 3, left). Thrombus aspiration was performed with aspiration catheter and red thrombi were retrieved. The residual stenosis of left anterior descending artery was mild after thrombus aspiration (Figure 3, right). Intravascular ultrasound virtual histology after thrombus aspiration showed calcified plaque without plaque rupture in culprit lesion, which might suggest embolic occlusion (Figure 4). Therefore, it was determined that stent insertion was unnecessary. It is assumed that embolization of cerebral vessels and coronary arteries occurred simultaneously or at least within a very short interval. Enoxaparin was immediately started and apixaban was administered 1 day later without any antiplatelet agent. Echocardiography revealed reduced left ventricular ejection fraction of 28%, and subsequent echocardiography at 2 months showed no change in left ventricular function.

DISCUSSION

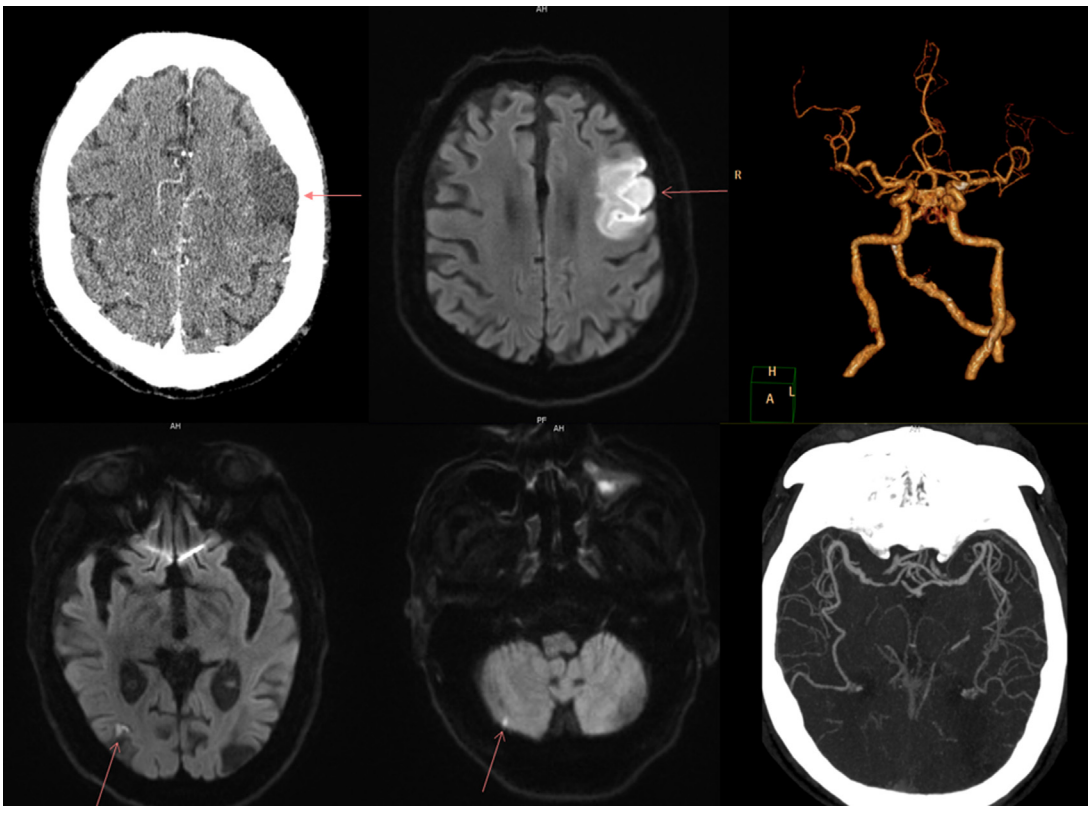
Epidemiologic data for cardiocerebral infarction (CCI) is very limited, with reported incidences ranging from 0.009% to 0.29% depending on the literature.[1-3] A recent meta-analysis revealed several characteristics of CCI.[4] The most common type of MI was anterior STEMI (38.3%), followed by inferior wall STEMI (27.7%). The most common culprit lesion in cranial arteries was MCA, with the left (30.9%) more prevalent than the right (19.1%). The most common causes

FIGURE 1 ECG Shows AF ST-Segment Elevation



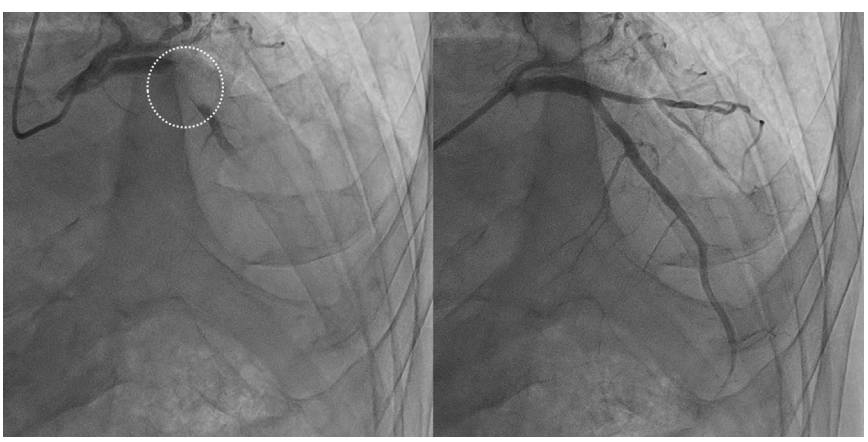
Twelve-lead electrocardiography (ECG) shows atrial fibrillation ST-segment elevation on V_2-V_6 and reciprocal change on leads III and aVF.

FIGURE 2 **CTA and MRI Show Cerebral Infarcts**



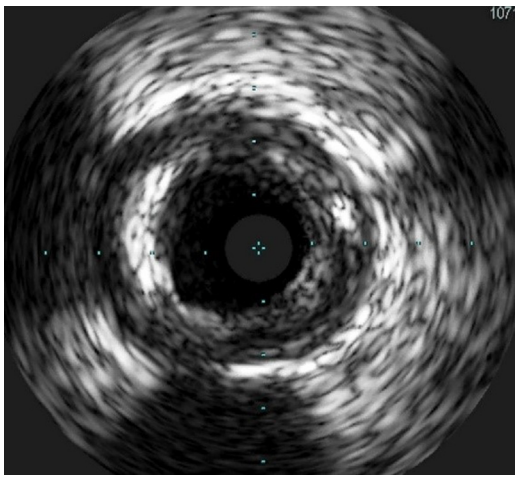
Brain computed tomography angiography (CTA) and magnetic resonance image (MRI) show multiple cerebral infarct (arrows).

FIGURE 3 **Coronary Angiography Before and After Thrombus Aspiration**



Coronary angiography shows total occlusion of the mid left anterior descending artery (left, dotted circle) and no significant stenosis in culprit lesion after thrombus aspiration, suggesting embolic occlusion (right).

FIGURE 4 IVUS Shows Calcified Plaque



Intravascular ultrasound (IVUS) shows calcified plaque without plaque rupture at culprit lesion.

of CCI were cardiogenic shock and heart failure (37.2%, respectively), and AF was 25.5%. In this case, anterior wall MI and left MCA infarction were observed.

The mortality rate at hospital discharge was 33.3%, and at 90 days, the mortality rate was 49.2%.[4] The majority of the patients passed away because of cardiac causes such as ventricle tachyarrhythmias, cardiac tamponade, aortic dissection, ventricle septal rupture, or sudden death. Approximately one-half of the patients exhibited neurologic deficit, followed by chest pain and/or dyspnea (53.2%). In contrast, symptoms of MI were the first to present in 19.1% of patients, followed by symptoms of acute ischemic stroke. MI and acute ischemic stroke symptoms were presented simultaneously in 26 patients (27.7%). In our case, the patient initially presented with neurological symptoms, followed by the identification of STEMI on electrocardiography.

The most accepted pathogenetic hypotheses for the occurrence of simultaneous or nearly simultaneous CCI are based on the following 3 mechanisms[5,6]:

1. Simultaneous thrombosis of the coronary and cerebral arteries, such as with AF, type I aortic dissection involving the coronary artery and common carotid artery,[7] or electrical injury resulting in coronary and cerebral artery spasm.[8]
2. Stroke caused by heart disease, such as intraventricular thrombosis, patent foramen ovale (complicated with right heart infarction), and cardiac shock after acute MI.
3. A cerebral-cardiac axis disorder or cerebral infarction can lead to myocardial injury. The insular cortex, which plays an important role in the regulation of the central autonomic nervous system, is related to AF, activation of cardiac sympathetic nerves, myocardial injury, and the interruption of circadian rhythms of blood pressure.

Due to the rarity and complexity of this condition, there are still limited recommendations or guidelines for its diagnosis and treatment. Hence the treatment of CCI is very individualized without uniformity. Ibekwe et al [9] reviewed 25 cases of CCI and found that 7 cases (28%) used mechanical thrombectomy, 15 (60%) underwent percutaneous coronary intervention, 9 (36%) received fibrinolysis with alteplase, and 12 (48%) had concurrent cardiac thrombus. Additionally, 16 cases (64%) reported outcomes, with 4 (16%) and 6 (24%) cases having modified Rankin Scale scores ≤3 at 1 and 3 months, respectively. The patient in this case arrived late to the hospital after the golden time, which is crucial for the direct care of acute stroke cases. In addition, the lesion located in a relatively peripheral vessel, such as M2, making it not an indication for mechanical thrombectomy. If the patient arrived at the hospital without delay, the NIHSS score would be 7, and tissue-type plasminogen activator infusion prior to the coronary angiography could be considered, leading to better neurologic and cardiologic recovery and prognosis. Therefore, it is necessary to educate the public on initial symptoms and signs of stroke, and family members should be comprehensively educated about the symptoms and signs of acute ischemic stroke to provide first aid and bring the patient to the hospital within the golden hour.

In our case, the patient's chest discomfort was not detected early due to global aphasia. The prominent neurologic symptoms focused the health care provider's attention on stroke evaluation, delaying the detection of MI. Percutaneous coronary intervention was performed, but the golden time for cardiac ischemia had passed, so revascularization could not recover cardiac function.

FOLLOW-UP

After rehabilitation, he was discharged after 83 days of hospitalization. He was admitted with acute decompensated heart failure 3 months later. Unfortunately, he died suddenly from asphyxia-complicated cardiac arrest during hospitalization.

CONCLUSIONS

We demonstrated a case of CCI associated with AF. Timely revascularization is crucial for CCI. CCI treatment should be tailored to the individual patient, given the lack of clear clinical guidelines available for treating this condition.

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KEY WORDS atrial fibrillation, myocardial infarction, stroke